ELSEVIER

Journal of Diabetes and Its Complications 16 (2002) 249–254

Journal of Diabetes and its Complications

Reversible acute renal failure and nephrotic syndrome in a Type 1 diabetic patient

Larissa Avilés-Santa[a,*], Robert Alpern[b], Philip Raskin[a]

[a]Division of Clinical Diabetes, Department of Internal Medicine, University of Texas Southwestern Medical Center at Dallas, 5323 Harry Hines Boulevard, G5.238, Dallas, TX 75390-8858, USA

[b]Division of Nephrology, Department of Internal Medicine, University of Texas Southwestern Medical Center at Dallas, 5323 Harry Hines Boulevard, G5.238, Dallas, TX 75390-8858, USA

Received 15 October 2001; accepted 9 November 2001

Abstract

Nephrotic syndrome is a condition commonly associated with end-stage renal disease secondary to diabetic nephropathy. It is usually associated with long-standing renal insufficiency, microalbuminuria, and overt proteinuria. We present a diabetic patient with acute oliguric renal failure and nephrotic syndrome. At presentation, he had a serum creatinine of 2.3 mg/dl, blood urea nitrogen (BUN) of 69 mg/dl, urinary protein excretion of 10.5 g/24 h, serum albumin of 1.3 g/dl, and a urine output <400 cc/24 h. A renal biopsy was done and the renal pathology was compatible with early diabetic nephropathy. Despite intense diuretic therapy, the patient's renal condition did not improve, and peritoneal dialysis was started several months after diagnosis. After 8 months of dialysis therapy, the patient's renal parameters and urinary output spontaneously restored to normal limits (serum creatinine was 1.1 mg/dl, urinary albumin excretion was 411 mg/24 h, serum albumin was 4.3 g/dl, and normal urine output) and dialysis was discontinued. His renal function did not deteriorate after discontinuation of dialysis. We conclude that this patient's reversible acute renal failure and nephrotic syndrome were associated with minimal change disease and not due to diabetic nephropathy. © 2002 Elsevier Science Inc. All rights reserved.

1. Introduction

Diabetic nephropathy is a slowly progressive condition usually preceded by a long-standing history of diabetes. Early signs of nephropathy are usually subclinical or incipient, and albuminuria is considered the hallmark of clinical disease. Nephrotic syndrome is usually seen at advanced stages of the disease. Renal failure and proteinuria associated with diabetic nephropathy are usually irreversible.

In this report, we present a patient with Type 1 diabetes mellitus since the age of 3 years, who experienced acute renal failure and nephrotic syndrome. After 8 months of dialysis therapy, his renal function and urinary albumin excretion reverted to normal parameters.

2. Case report

RH, a 28-year-old non-Hispanic White man, was admitted to the University Diabetes Treatment Center at Parkland Memorial Hospital on December 29, 1995, with a chief complaint of progressive peripheral edema and decreased urinary output. The patient noticed swelling of his ankles for the first time 1 month prior to admission. Gradually, the swelling involved the legs, and his urine output decreased. He denied pain, swelling or redness of the joints, shortness of breath, sore throat, cough, exertional dyspnea, paroxysmal nocturnal dyspnea, hemoptysis, fever, chills, or skin rashes. He also denied use or abuse of nonsteroidal antiinflammatory agents or any over-the-counter drugs.

On December 19, 1995, he sought medical advice for these problems at an ambulatory care facility. Blood chemistries revealed a serum creatinine level of 1.1 mg/dl and a blood urea nitrogen (BUN) level of 36 mg/dl. Oral loop diuretics were started. On December 27, 1995, the patient returned to the clinic for a follow-up visit. He stated that the swelling had become generalized, his scrotum was

* Corresponding author. Tel.: +1-214-648-2017; fax: +1-214-648-4854.

1056-8727/02/$ – see front matter © 2002 Elsevier Science Inc. All rights reserved.
PII: S1056-8727(01)00221-5

extremely swollen, and he had almost no urine output. Blood chemistries revealed a serum creatinine of 1.9 mg/dl and BUN of 62 mg/dl. The dose of oral diuretics was increased. On December 29, 1995, the patient came to the emergency room because of persistent swelling, 40 lb of weight gain, and minimal urine output.

RH had Type 1 diabetes mellitus since the age of 3 years, nonproliferative diabetic nephropathy, hypothyroidism since the age of 12, and Charcot–Marie–Tooth peripheral neuropathy 1 year prior to admission. He also had nonproliferative diabetic retinopathy. His medications consisted of NPH insulin 20 U before breakfast and 15 U before supper and levothyroxine 125 μg/day. The patient had not been hospitalized for diabetes since age 15, although he failed to keep several medical appointments and often omitted doses of his medications.

On physical examination, the patient weighed 80.3 kg, and his blood pressure was 140/90 mm Hg. He was not in acute distress. He had anasarca with pitting edema of the abdominal wall, both upper and lower extremities, the scrotum, and penis. He did not have xanthomas or xanthelasmas, and the thyroid gland was not palpable. He had absent breath sounds at both lower pulmonary fields. He did not have murmurs, gallops, or rubs. A urine analysis yielded gross proteinuria and oval fat bodies. No red or white blood cells were observed. His serum creatinine was 2.3 mg/dl and his BUN was 69 mg/dl. Acute oliguric renal failure was diagnosed and the patient was hospitalized. Oral furosemide was administered and the dose progressively increased 200 mg orally every 6 h. He had a urine protein excretion of 10.5 g/24 h. Other laboratory tests yielded a hemoglobin A_{1c} (HbA_{1c}) of 7.7% (range for nondiabetic individuals <5.6%), a free thyroxine level of 1.0 ng/dl, a total T3 of 33.0 ng/dl, free T3 index of 38, a TSH greater than 150 μU/ml, and total plasma cholesterol of 881 mg/dl (Table 1). Further workup for acute renal failure and proteinuria included antinuclear antibodies (absent), hepatitis A IgG and IgM (negative), hepatitis B surface and core antigens (negative), hepatitis B surface antibody (nonreactive), and hepatitis C antigen (nonreactive). Anti-streptolysin O titers, antineutrophil cytoplasmic antibodies, erythrocyte sedimentation rate, complement 3 and 4 levels, and rheumatoid factor levels were not measured.

A renal sonogram revealed kidneys of normal size without hydronephrosis. A renal biopsy was performed (Fig. 1). Fifteen glomeruli were identified in the biopsy specimen. There was diffuse expansion of the mesangial matrix and focal intercapillary sclerosis. Immunofluorescence techniques yielded negative staining, except IgG of the glomerular basement membrane, consistent with diabetic nephropathy.

Table 1
Profile of laboratory test results

Date	Serum creatinine (mg/dl)	BUN (mg/dl)	Serum albumin (g/dl)	Serum total protein (g/dl)	Urine protein excretion (mg/24 h)	Urine albumin excretion (mg/24 h)	HbA_{1c} (%)	TSH (μU/ml)	Free T4e (ng/dl)	T-Chol (mg/dl)	HDL-C (mg/dl)	LDL-C (mg/dl)	TG (mg/dl)
12/19/95	1.1	36											
12/27/95	1.9	62											
12/29/95	2.3	69	1.3	4.7	10,532		7.8	>150	1.0	878	65	770	215
1/1/96	3.2	85											
1/7/96										547	44	427	381
1/8/96								133.4	0.9				
1/10/96	2.4	98	<1.0										
1/18/96	1.6												
1/25/96	1.5												
2/1/96	1.4							>150	0.9				
2/15/96	2.3	52	2.1	4.2			7.1			325	56	208	305
2/22/96	2.0		2.1	4.2	9528	746	7.0			393			326
3/7/96	2.5	61	1.9	3.8			6.9	>150	0.9	495	58	207	547
3/14/96	2.2	55	2.0										
4/4/96	2.1	44	2.0	3.8			6.9	64.2	1.1	869	63	278	435
5/16/96	3.1	43	1.8	3.6			6.4	50.0	0.9	1196	56	348	575
6/25/96	4.0	47	<1.0		15,729	1713	5.9	13.8	1.1	970	42	294	442
7/23/96	5.4												
8/8/96							6.6	>150	0.4	967	59	360	633
10/03/96	3.9	40	2.3	4.8			8.3			863	107	699	287
11/15/96								>150	0.6				
11/22/96							8.2		0.4	896	105	322	409
2/20/97	1.3	29	2.5	5.4			8.5	>150	1.1	803	80	325	304
4/3/97	1.3	30	2.9	6.2									
6/17/97			4.1	6.8									
11/21/97	1.1		4.3				10.1	143.2	1.1	233	80	126	134
12/4/97					411								
3/6/98							9.9	148.3	0.9	278		155	140

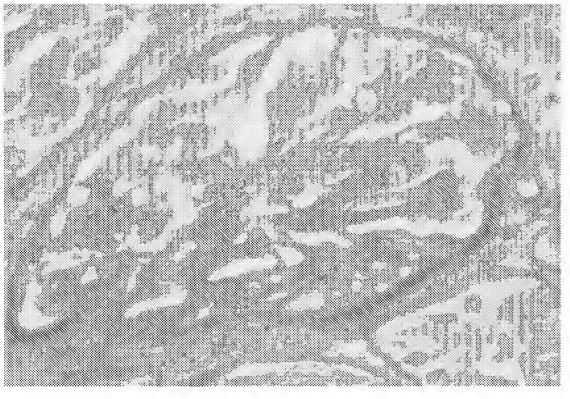

Fig. 1. Light microscopic appearance of one of the H–E stained renal biopsy specimens. (Magnification 100X).

Oral diuretic therapy did not improve the patient's urine output nor reduce his anasarca. Thus, an intravenous infusion of furosemide and mannitol was administered in combination with oral metolazone and chlorothiazide. Oral thyroid hormone replacement was increased, but poor gastrointestinal absorption secondary to bowel wall edema was suspected. Therefore, intravenous levothyroxine was administered at a dose of 150 μg/day. Lovastatin 80 mg/day was given to lower plasma cholesterol levels.

After starting the intravenous diuretic infusion, the patient's body weight progressively decreased from 80.3 kg on Day 1 to 61.6 kg on Day 12, with a subsequent improvement in his peripheral and scrotal edema. His serum creatinine did not change during his hospital stay. His urinary output increased. Serum-free thyroxine levels did not change, and TSH levels decreased to 133.4 μU/ml. Intravenous levothyroxine was discontinued on Day 10, and oral levothyroxine at a dose of 125 μg/day given instead.

The patient was discharged on Day 13. On discharge, his medications consisted of furosemide 200 mg bid, metolazone 10 mg QD, NPH 15 U sq QD before breakfast, levothyroxine 125 μg QD, lovastatin 40 mg QD, potassium chloride 10 meq QD, and calcium carbonate 500 mg tid.

The patient was seen at 2-week intervals after discharge. TSH remained elevated above 150 μU/ml, whereas free thyroxine stayed within the low normal range. Serum creatinine levels decreased to a range of 1.1–1.5 mg/dl during the next 8 weeks following discharge. The dose of diuretics was slowly increased (furosemide 200 mg tid–qid and metolazone 10 mg qid). The edema slowly decreased but did not completely resolve.

Five months after discharge (June 1996), the patient experienced recurrence of the edema and weight gain. His serum creatinine levels increased to 5.2 mg/dl. He was readmitted, and intravenous diuretics were administered without improvement of the renal function. He was discharged on metolazone 10 mg qid, ramipril 5 mg QD, furosemide 200 mg bid, simvastatin 40 mg HS, Epogen 3000 U sc every Monday, Wednesday, and Friday, ferrous gluconate 300 mg qid, heparin 5000 U bid, levothyroxine 100 μg QD, and calcium acetate 2 tablets qid. Continuous ambulatory peritoneal dialysis (CAPD) was started in July 1996.

The patient had a stable medical course throughout the following year while on CAPD. In the ensuing months, his peripheral edema progressively disappeared, his urine output and creatinine clearance increased to normal parameters, his total cholesterol and lipoprotein levels decreased to normal values, and his blood chemistries improved. CAPD was stopped and his peritoneal cannula was removed in February 1997. After discontinuation of CAPD, the patient's renal and volume status remained within the normal range, and he no longer required oral diuretics, iron, or Epogen. He was seen again at the Diabetes Clinic in November 1997. He denied any complaints. His blood pressure was 112/84. He was taking insulin and levothyroxine erratically. His laboratory test results revealed a serum creatinine of 1.1 mg/dl, a HbA_{1c}

of 10.1%, a TSH of 143.2 μU/ml, free T4c of 1.1 ng/dl, and high total cholesterol (233 mg/dl) and LDL-C (126 mg/dl) levels. His protein albumin excretion, measured 2 weeks later, was 411 mg/24 h. Simvastatin and quinapril were restarted, and his insulin dosage adjusted in order to achieve normoglycemia. At a follow-up visit on March 18, 1998, his thyroid function tests, lipoprotein profile, and HbA_{1c} were similar to those measured in November 1998. He has not returned to the clinic since.

3. Discussion

The sudden onset and rapid progression of renal failure coupled with the extraordinary plasma lipid abnormality originally attracted our attention to the patient. However, his subsequent course with almost unexplainable reversal of almost all the abnormalities at presentation became the most outstanding feature of this case.

The course of diabetic nephropathy in Type 1 and Type 2 diabetic individuals was probably first described by Krowleski and Fabre, respectively (Fabre, Balant, Dayer, Fox, & Vernet, 1982; Krowlewski, Warram, Chritlieb, Busick, & Kahn, 1985). Krowlewski et al. (1985) observed that the risk for persistent proteinuria in Type 1 diabetic individuals increased rapidly between the fifth and fifteenth year of duration of diabetes. They also observed a direct relationship between glycemic control during the first fifteen years of diabetes and the risk for renal disease. Fabre et al. (1982) observed that the interval between overt proteinuria and end-stage renal disease could be 5 years or shorter in Type 2 diabetic individuals (Krowlewski et al., 1985). This observation could be explained by the fact that the onset of Type 2 diabetes sometimes precedes its diagnosis for several years, and microvascular complications are present at the time of diagnosis in about 20% of individuals. More recent observations have better characterized the course of diabetic nephropathy. Microalbuminuria, for example, has been identified as the predictive factor for the development of overt proteinuria (Lapuz, 1997). Microalbuminuria precedes the onset of overt proteinuria and raise in serum creatinine and urea nitrogen levels. Therefore, diabetic nephropathy is a slowly progressive, irreversible disease, characterized by an asymptomatic course during the first 5–10 years of diabetes, followed by microalbuminuria, preceding the onset of overt proteinuria, renal insufficiency, and, finally, end-stage renal disease. Abrupt deterioration in renal function is not characteristic of diabetic nephropathy (Lapuz, 1997), with the exception of that seen occasionally during pregnancy (Kitzmiller & Combs, 1996; The Diabetes Control and Complication Trial Research Group, 1993).

Adequate diabetes and blood pressure control are determining factors in the progression of diabetic renal disease (Fabre et al., 1982; Krowlewski et al., 1985; Reece, Leguizamon, & Homko, 1998). Maintenance of normoglycemia and control of arterial blood pressure delay the progression of diabetic nephropathy (Lapuz, 1997; Reece et al., 1998). The use of angiotensin-converting enzyme inhibitors also has been shown to delay the progression of diabetic nephropathy in either hypertensive or normotensive patients with microalbuminuria (Hebert et al., 1994; Wilmer et al., 1999).

The sudden onset of our patient's symptoms and clinical signs suggested an acute renal insult other than diabetes as the etiology of his condition. Despite the patient's long-standing history of diabetes, abnormal renal function or hypertension had not been identified prior to his admission in December 1995. This patient did not show the characteristic course of diabetic nephropathy prior to hospital admission. The acute elevation in serum creatinine and BUN, the sudden onset of peripheral edema, and the decreased urinary output suggested an etiology other than diabetic nephropathy. Nevertheless, no other obvious cause for acute renal failure was identified.

Marked hypothyroidism was considered the precipitating factor of this patient's acute nephrotic syndrome. This was obviously incorrect because, at follow-up at the clinic, the patient's TSH levels decreased almost to normal, but there was no improvement in renal function. The patient's free thyroxine levels reached normal levels, but the TSH levels continued to be elevated. Thyroid hormone resistance was suspected, but no evidence of a previous history of intractable hypothyroidism (high TSH levels) and the observed decrease in TSH levels with oral levothyroxine replacement did not support this view. Inadequate intake of levothyroxine is probably the explanation for this patient's persistent thyroid function abnormalities.

A renal biopsy was done in order to make a diagnosis. The biopsy revealed sclerotic changes compatible with diabetic nephropathy. Immune staining did not reveal any immune complex deposits as can be seen in glomerulopathies secondary to collagen vascular diseases or infectious etiologies. Thus, it seemed clear that this patient had a rapidly progressive exacerbation of diabetic nephropathy leading to renal failure and the need of renal replacement therapy. After his second hospitalization, it was thought that his renal dysfunction was irreversible, and dialysis therapy was started. However, during the course of peritoneal dialysis treatment, the patient experienced a progressively spontaneous improvement of his peripheral edema, lipid abnormalities, and renal function, and CAPD was discontinued.

Acute renal failure, although uncommon, has been observed in patients with nephrotic syndrome and minimal change disease, without previous history of renal disease (Chamberlain, Pringle, & Wrong, 1966; Holdsworth, Stephenson, Dowling, & Atkins, 1977; Hulter & Bonner, 1980; Jennette & Falk, 1990; Smith & Hayslett, 1992; Tinawi, Salinas-Madrigal, & Domoto, 1995). Chamberlain et al. (1966) were the first to describe this phenomenon. In 1966, this group described nine patients with acute, oliguric renal failure associated nephrotic syndrome. Four of these patients had minimal change nephropathy, and in two cases,

the acute renal failure reversed after hemodialysis and ultrafiltration treatment.

Reversal of acute renal failure associated with acute nephrotic syndrome and minimal change disease has been described by other groups (Holdsworth et al., 1977; Hulter & Bonner, 1980; Jennette & Falk, 1990; Koomans, Hene, & Dorhout Mees, 1992; Loghman-Adham, Siegler, & Pysher, 1997; Lowestein, Schacht, & Baldwin, 1981; Scully, Mark, & McNeely, 1982; Sjoberg, McMillan, Bartram, & Copley, 1983; Smith & Hayslett, 1992; Stephens, Yates, Lechler, & Baker, 1979; Tinawi et al., 1995). Reversal of acute renal failure has been observed after variable courses of dialysis (Holdsworth et al., 1977; Jennette & Falk, 1990; Lowestein et al., 1981; Scully et al., 1982; Sjoberg et al., 1983), and reversal of nephrotic syndrome has occurred after steroid therapy (Holdsworth et al., 1977; Jennette & Falk, 1990; Lowestein et al., 1981; Scully et al., 1982; Sjoberg et al., 1983) or spontaneously (Holdsworth et al., 1977). On the other hand, Hulter and Bonner (1980) and Raij, Keane, Leonard, and Shapiro (1976) did not observe improvement in either renal function or urinary albumin excretion after dialysis therapy despite prolonged follow-up.

Reversibility of nephrotic-range proteinuria caused by diabetic nephropathy has only been observed and reported by the Captopril Study Group (Hebert et al., 1994; Wilmer et al., 1999). In their first report (Hebert et al., 1994), remission was observed in 16.7% of patients with nephrotic syndrome receiving captopril. The baseline urinary protein excretion was 5.2 g/24 h, and 3.4 years later, the urinary protein excretion was 1.5 g/24 h. In a more recent follow-up report (Wilmer et al., 1999), those patients have stayed in remission for 7.7 years with stable urinary protein excretion (1.03 g/24 h). Spontaneous remission of nephrotic syndrome associated with diabetic nephropathy has not been reported.

The etiology of acute renal failure associated with minimal change disease is unknown. Interstitial edema (Chamberlain et al., 1966; Hulter & Bonner, 1980; Logham-Adham et al., 1997; Lowestein et al., 1981; Scully et al., 1982; Sjoberg et al., 1983; Tinawi et al., 1995) and acute tubular necrosis (Chamberlain et al., 1966; Jennette & Falk, 1990; Logham-Adham et al., 1997; Tinawi et al., 1995) have been observed in renal biopsies. A proposed mechanism for the acute renal failure is renal interstitial edema with resultant tubular obstruction, increased hydrostatic pressure in Bowman's space, and decreased glomerular filtration rate (Lowestein et al., 1981; Scully et al., 1982). Other investigators have suggested an increased renal susceptibility to ischemia (Hulter & Bonner, 1980) as the mechanism for renal injury.

Our patient had most of the clinical features shared by the patients previously reported, he did not have underlying renal disease prior to acute renal failure, and his course was characterized by hypertension, oliguria, and a short duration of the nephrotic syndrome preceding the acute renal injury, as described in all patients (Holdsworth et al., 1977; Hulter & Bonner, 1980; Scully et al., 1982; Sjoberg et al., 1983; Smith & Hayslett, 1992). However, our patient was much younger than patients previously described (mean age is 58–60 years) (Holdsworth et al., 1977; Hulter & Bonner, 1980; Smith & Hayslett, 1992).

Like in other reports, our patient showed a similar clinical course of peripheral edema and nephrotic syndrome preceding acute renal failure, complicated by oliguria and hypertension. However, the renal biopsy did not reveal interstitial edema as in some of the reports. In addition, in some of the reports, dialysis seemed to have helped restore intravascular and intraglomerular volume, although, not changing the course of the disease.

In conclusion, our patient's clinical course suggests that he had underlying glomerular basement changes compatible with diabetic nephropathy that were not the sole etiology of his acute renal failure and nephrotic syndrome. Minimal change disease was most likely the etiology of this patient's condition.

References

Chamberlain, M. J., Pringle, A., & Wrong, O. M. (1966). Oliguric renal failure in the nephrotic syndrome. *Quarterly Journal of Medicine, 138*, 215–235.

Fabre, J., Balant, L. P., Dayer, P. G., Fox, H. M., & Vernet, A. T. (1982). The kidney in maturity onset diabetes mellitus: a clinical study of 510 patients. *Kidney International, 21*, 730–738.

Hebert, L. A., Bain, R. P., Verme, D., Cattran, D., Whittier, F. C., Tolchin, N., Rohde, R. D., Lewis, E. J., Collaborative Study Group. (1994). Remission of nephrotic range proteinuria in type 1 diabetes. *Kidney International, 46*, 1688–1693.

Holdsworth, D., Stephenson, P., Dowling, J., & Atkins, R. (1977). Reversible acute renal failure in the nephrotic syndrome with minimal glomerular pathology. *Medical Journal of Australia, 2*, 532.

Hulter, H. N., & Bonner, E. L. (1980). Lipoid nephrosis appearing as acute renal oliguric renal failure. *Archives of Internal Medicine, 140*, 403–405.

Jennette, J. C., & Falk, R. J. (1990). Adult minimal change glomerulopathy with acute renal failure. *American Journal of Kidney Diseases, 16*, 432–437.

Kitzmiller, J. L., & Combs, C. A. (1996). Diabetic nephropathy and pregnancy. *Obstetrics and Gynecology Clinics of North America, 23* (1), 173–203.

Koomans, H. A., Hene, R. J., & Dorhout Mees, E. J. (1992). Acute renal failure complicating nephrotic syndrome. In: S. Cameron, A. M. Davison, J. F. Grunfeld, D. Kerr, & E. Ritz (Eds.), *Oxford textbook of clinical nephrology* (1st ed., pp. 1034–1040). Oxford, UK: Oxford University Press.

Krowlewski, A. S., Warram, J. H., Christlieb, A. R., Busick, E. J., & Kahn, C. R. (1985). The changing natural history of nephropathy in type 1 diabetes. *American Journal of Medicine, 78*, 785–794.

Lapuz, M. H. S. (1997). Diabetic nephropathy. *Medical Clinics of North America, 81* (3), 679–688.

Loghman-Adham, M., Siegler, R. L., & Pysher, T. J. (1997). Acute renal failure in idiopathic nephrotic syndrome. *Clinical Nephrology, 47* (2), 76–80.

Lowestein, J., Schacht, R. G., & Baldwin, D. S. (1981). Renal failure in minimal change nephrotic syndrome. *American Journal of Medicine, 70*, 227–233.

Raij, L., Keane, W., Leonard, A., & Shapiro, F. (1976). Irreversible acute

renal failure in idiopathic nephrotic syndrome. *American Journal of Medicine*, *61*, 207.

Reece, E. A., Leguizamon, G., & Homko, C. (1998). Pregnancy performance and outcomes associated with diabetic nephropathy. *American Journal of Perinatology*, *15* (7), 413–421.

Scully, R. E., Mark, E. J., McNeely, B. U. (Eds.) (1982). Case records of the Massachusetts General Hospital (Case 4-1982). *New England Journal of Medicine*, *304*, 221.

Sjoberg, R. J., McMillan, V. M., Bartram, L. S., & Copley, J. B. (1983). Renal failure with minimal change nephrotic syndrome: reversal with hemodialysis. *Clinical Nephrology*, *20* (2), 98–100.

Smith, J. D., & Hayslett, J. P. (1992). Reversible renal failure in the nephrotic syndrome. *American Journal of Kidney Diseases*, *19* (3), 201–213.

Stephens, V., Yates, A., Lechler, R., & Baker, L. (1979). Reversible uremia in normotensive nephrotic syndrome. *British Medical Journal*, *2*, 705.

The Diabetes Control and Complication Trial Research Group. (1993). The effect of intensive insulin treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *New England Journal of Medicine*, *329* (14), 977–986.

Tinawi, M., Salinas-Madrigal, L., & Domoto, D. (1995). Minimal change disease presenting with acute tubular necrosis. *American Journal of Kidney Diseases*, *25* (4), 648–650.

Wilmer, W. A., Hebert, L. A., Lewis, E. J., Rohde, R. D., Whittier, F., Cattran, D., Levey, A. S., Breyer Lewis, J., Spitalewitz, S., Blumenthal, S., & Bain, R. P. (1999). Remission of nephrotic syndrome in type 1 diabetes: long-term follow-up of patients in the Captopril study. *American Journal of Kidney Diseases*, *34* (2), 308–314.